THE INVESTIGATION OF PERSISTENT DIARRHOEA

BY

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Diarrhoea is the passage of loose stools so frequently as to cause annoyance. To some patients, four bowel actions a day is an intolerable nuisance, some are much more stoical, while others complain freely when the inconvenience is less. The severity of pain, urgency and tenesmus accompanying the diarrhoea often determines whether a patient seeks medical help or not. By persistent diarrhoea is meant the occurrence of frequent loose stools, continually or intermittently for a period of over three weeks.

It is true that many patients with chronic diarrhoea begin their illness abruptly, but the problem of acute flux itself is beyond the scope of this paper. Whereas in acute cases the emphasis is on the search for and treatment of an infective agent, together with the palliation of symptoms and treatment of dehydration, in the case of persistent diarrhoea the search for a cause has spread far beyond the confines of infection. Indeed, in temperate climates an infective cause is the exception rather than the rule. Yet the almost automatic action on the part of many doctors faced with a case of persistent diarrhoea is to obtain a specimen of faeces and send it many miles to a public health laboratory with "? *organisms*" on the request card. The patient's difficulties with the ice cream carton provided are matched by those of the bacteriologist at the other end, who naturally finding plenty of organisms has to report in such terms as "*No significant departure from the normal bowel flora*". In circumstances like these it is well to ask what precisely we are looking for, and I suppose the answer would be "typhoid and paratyphoid, and things like that". It is true that in infants and children, cases of paratyphoid fever may occur with persistent diarrhoea as a main symptom, but in the adult, diarrhoea if it occurs at all is completely overshadowed by the feverish and constitutional symptoms. It is true also that in the very old or debilitated, chronic Sonne Dysentery or other salmonella infections may cause persistent diarrhoea. In these cases, especially if the illness is accompanied by fever, the bacteriological search for non-lactose fermenting organisms by culture is worthwhile.

Where the patient has been abroad and chronic amoebic dysentery or lambliasis are suspected the examination of old stale specimens of faeces is completely useless. If these diseases are suspected the faeces should be taken straight from the rectum to the microscope slide, preferably after a sigmoidoscopic examination, but failing that a fresh stool should be taken straight to the laboratory in the bed pan, and the search for parasites begun at once. Worms do not give rise to diarrhoea in this country.

Many man-hours and money are wasted on fruitless pathological examinations of faeces. It is a form of long distance investigation that could well be dispensed with. In fact, the diagnosis of continuing diarrhoea is much more direct, though it is not simple. What is required is a finger stall or glove, a good illuminated proctoscope or small sigmoidoscope, some powders or tablets for testing for occult blood, and a syringe to withdraw some blood for the estimation of haemoglobin and E.S.R. at the nearest laboratory. Other investigations may be necessary, but one can get a long way with the simple procedures.

Table I gives a list of the commoner disorders which give rise to persistent diarrhoea, and these are arranged in main groups according to the basic mechanism and pathology. Table II gives the expected findings in each group with each of the main investigatory procedures.

TABLE I

CLASSIFICATION OF CAUSES OF PERSISTENT DIARRHOEA

Main Groups	Varieties
Nervous and functional ..	"Lienteric". "irritable colon syndrome". "Mucous colitis." Episodic with vomiting. Associated with general diseases.
Malabsorbtion	"Sprue". Coeliac Disease. Idiopathic steatorrhoea. Pancreatitis. Loop syndrome. After gastrectomy. Hepatogenous.
Inflammatory	Ulcerative colitis. Proctitis. Ileitis. Ileo-colitis. Diverticulitis.
Infective	Amoebic dysentery. Chronic dysentery. Lambliasis.
Neoplastic..	Carcinoma of colon (commonly caecum or rectum). Rare tumours of the small intestine.

Rectal examination—one cannot overstress the importance of making a digital rectal examination in all patients with chronic diarrhoea. The fact that carcinoma of the colon is one of the most operable of all carcinomas and that 30 per cent of colonic carcinomas occur in the rectum should stimulate us to make this essential if unpleasant examination. Small finger stalls which are easily disposable are, outside Hospitals, more practicable than the flanged, heavier type. If carcinoma is suspected the finger should explore as high as possible, and bimanual examination be attempted.

Occult blood tests—there are now a variety of tablets and powders which when dissolved in glacial acetic acid and poured onto a blood-containing faecal smear on paper or glass give rise to a dark blue or green colouration. Gregerson's powder is no longer obtainable because the makers will not handle Benzidine which has been proved to be carcinogenetic in animals. An alternative powder is made up of Barium Peroxide 0.2 g. and Ortho Tolidine 0.025 g. "Hematest" tablets can also be used for testing a faecal smear by placing the tablet on the faecal smear and moistening the tablet and smear with two drops of water. A strongly positive occult blood test is nearly always significant of intestinal bleeding, though it must be remembered that the bleeding can be from the nose, the gums, or the anal canal. The test is not positive in the presence of iron, but intestinal hurry may cause haemoglobin rich animal meat fibres to appear in the stool and give a weakly positive (green) reaction.

Two or three successive positive tests are more significant than a single positive test, but the finding of a negative test is strong evidence against carcinoma or ulcerative colitis. In this way the rectal examination and subsequent occult blood test of a faecal smear obtained from the examining finger stall or glove provide a useful "screening" procedure, negative findings tending to promote a sense of security and positive finding being regarded as a call for immediate further investigation.

Proctoscopy and sigmoidoscopy—in out-patient practice the rectum only is inspected. A simple small, proximally lighted sigmoidoscope is the best instrument. The presence of faeces may prevent a careful examination of the upper rectum, but they rarely conceal the appearance of the rectal mucosa. In 95 per cent of cases of ulcerative colitis the mucosa has a very typical reddened, granular appearance. The veins so visible in normal mucosa, cannot be seen in cases of ulcerative colitis. The mucosa also bleeds easily and ulcers may be seen, though the last finding is not essential for a diagnosis.

Amoebic ulcers are small irregular and pitted, surrounded by a rim of red mucosa but beyond this all is normal.

The appearances of malignancy vary: there is rarely any doubt as to the diagnosis in this country, though infective granulomas may be a source of difficulty in the tropics.

Stool appearance—the patients description of the frequency, and appearance of the stools is valuable evidence, but the appearance of the stool at sigmoidoscopy, or at

TABLE II

THE FINDINGS IN THE MAIN GROUPS OF CAUSES OF PERSISTENT DIARRHOEA

Main Groups	P.R.	Occult blood	Sigmoidoscopy	Stool appearance	E.S.R.	Hb.	Barium enema	Daily fat excretion in stools
Nervous and functional ..	Nil	—	Nil (or excess mucous)	Varies	Normal	Normal	? spasm	Normal
Malabsorbtion	Nil	—	Nil (mild hyperaemia sometimes)	Fatty	Normal	Low	Normal	Over 5 gms.
Inflammatory	? granular mucosa	+	Diagnostic in 95 per cent of ulcerative colitis and proctitis	Offensive blood ±	Much raised	Low	Usually abnormal	Usually normal
Infective	Nil	±	? amoebic ulcers	Offensive blood ±	?	?	May be normal	Normal
Neoplastic..	? mass	+	? mass or blood from above	Offensive blood ±	Raised	Low	Filling defect seen	Normal

inspected in the bed pan may give the clue to diagnosis. The stool in the sprue–like malabsorbtion syndromes is bulky, pale and greasy in appearance. Often it is frothy. The presence of mucus is not to be taken as alarming, for this is a common finding in many functional disorders of the intestine. The presence of visible blood coming from above the anal canal is of immense significance.

E.S.R. and haemoglobin—these are rarely normal in ulcerative colitis and Crohn's Disease. In malabsorbtion states, the haemoglobin may be low and the E.S.R. normal. The same applies to carcinoma of the colon. These tests are simple to perform in a laboratory, or in the clinic, and as with the occult blood test they are most useful as a "screening" or preliminary procedure.

Barium enema—this is often the only way of demonstrating disease of the caecum or terminal ileum whether it be inflammatory or neoplastic. In ulcerative colitis, a barium enema tells something of the extent of the disease and the severity of mucosal involvement. As a routine procedure for the exclusion of carcinoma of the colon it is not recommended unless the limitations of the method are fully understood. A negative report does not mean that a carcinoma is not present, it merely means that with the relatively crude methods available, a carcinomatous filling defect has not been seen. Faeces still present after bowel lavage are the greatest source of difficulty in interpretation. In general, a barium enema should not be done until all the other investigations have been made and evaluated, and the report should be placed in perspective.

Enfoliative Cytology—no really satisfactory technique for the diagnosis of colonic carcinoma using this method has been devised and, though many examinations are made in America, it is not considered a practical proposition over here.

Assessment of probabilities—in the end one comes back to clinical judgement in all problems of diagnosis, and this is particularly true when considering a case of persistent diarrhoea. So far the mechanical, chemical and haematological aids to diagnosis has been stressed with a rather different clinical approach to this problem because ordinary "routine" medical examination yields so few clues in this type of case. Nevertheless, at the end of all investigations, there is often a need to reach an opinion based on conflicting evidence; or on insufficient understanding of the pathological and physiological factors controlling intestinal function. So little is known about the mechanism of chronic diarrhoea: why, for example, one patient with ulcerative colitis will pass twenty stools a day, whereas another with the disease apparently of the same extent and severity, passes two stools a day. And so little is known about the mechanisms behind so many of the functional bowel disorders—those distressing conditions where the patients seem to have lost all the natural rhythym of their intestinal function. Some cases of diarrhoea last three to six months, and then clear spontaneously for no known reason. Sometimes in these cases the stools may appear fatty and actually contain excess fat, and it is clear that the disorder is of the small intestine or pancreas, or both; it seems, however, to be self limiting. In other rather similar cases, the rectal mucosa may appear hyperaemic, and one is inclined to think in terms of an allergic basis. However there is no doubt that it is to the patients' benefit if an early diagnosis is made of ulcerative colitis, or carcinoma of the colon, and that appropriate treatment can alleviate the troubles of those with malabsorbtion syndromes, or of those with functional diarrhoea.

"*Functional Diarrhoea*"—this subject requires a special note because the diagnosis is based not only on the negative results of investigations, but also on the pattern of symptoms and the patient's general history, physical constitution and behaviour.

Certain symptom complexes occur more frequently than others. The so-called Lienteric Diarrhoea which is an exaccerbation of the normal gastro-colic reflex, is rarely a cause for concern, but may sometimes cause alarm to an anxious parent, or an anxious patient. Reassurance and explanation do much good.

The irritable colon syndrome—alias spastic colon, is characterised by the loss of any regular bowel habit. Phases of constipation are often succeeded by violent diarrhoea and sometimes the latter becomes a habit. Small hard, or normal stools, surrounded

by copious mucous are often passed. One variant is the badly named "*mucous colitis*" where an irritable rectum constantly desires to empty itself of small quantities of mucous which has been produced by over-active, over-stimulated goblet cells.

There are also sufferers from an over-sensitive rectum, whose urgent defaecation becomes a habit whenever they have to keep an appointment or leave the house. The trouble becomes a habit and an obsession, interfering with and modifying their lives. It is often associated with pruritus, strangury and genital symptoms. The basis may be emotional, but one sometimes feels that some of the sufferers are unlucky to have such a poor control apparatus in their sacral autonomic system.

Episodic Diarrhoea—some patients are subject to violent bouts of diarrhoea, often with vomiting, which last from twelve to forty-eight hours and which occur at intervals of one to six months. The disturbance may be akin to migraine or epilepsy—a sort of autonomic nerve storm. Nothing is known of the pathology or underlying mechanism. The sufferers are often nervous subjects prone to asthma, migraine or eczema.

SOME MODERN DEVELOPMENTS IN PERIPHERAL VASCULAR SURGERY

BY

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One of the most important advances in arterial surgery has been the advent of direct operations upon arteries including the use of various forms of grafting, and these have partly displaced operations on the sympathetic nervous system. The largest field for this work is the ever increasing problem of atherosclerosis but no less important, though less common, is the place of direct surgery in patients with injuries involving arteries.

Modern arterial surgery requires an artery bank, and some form of artery bank has been in existence in Bristol since 1952. In a few cases the material that has been used for grafts has been autogenous vein or a plastic prosthesis, but homologous artery has proved to be by far the most suitable material and, until recently, the segments have been taken in the post-mortem room under sterile conditions and stored until needed. Full details of this artery bank and a new method for sterilizing artery segments is to be the subject of a separate communication elsewhere; a summary of the advantages and disadvantages of the various materials used in grafting are given in Table I.

TABLE I

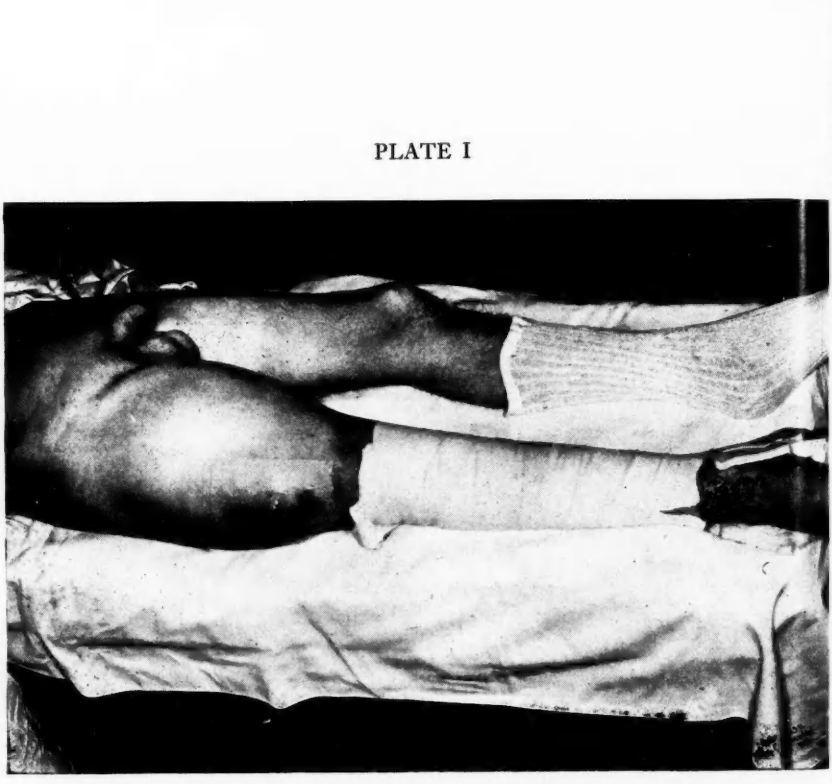
The advantages and disadvantages of various materials used in grafting.

	Advantages	Disadvantages	Indication
Homograft	Ease of handling. Good long term results.	1. Need for bank. 2. Supply difficulty. 3. Growth does not take place.	Any form of arterial replacement.
Heterograft	Availability.	High incidence of rupture.	NONE.
Venous autograft	Availability. Cells of graft survive.	Dilatation when not supported. Only short length available.	Grafting in thigh where muscle or fascial support available. Short lengths e.g. Trauma.
Prosthesis	Availability.	High incidence of thrombosis in arteries under 6 mm diameter	Replacement proximal to inguinal ligament.

ARTERIAL TRAUMA

War provides much experience in the surgical management of trauma, and the first serious attempts to restore damaged arteries were made in World War II. Although sporadic attempts to treat arterial injuries by suture had been made since the time of Carrel's classic work fifty years ago, the standard treatment for many years was

PLATE I



False aneurysm of the profunda femoris artery associated with a fracture of the femur.

ligature of the artery proximal to the site of injury. Ligation of the common femoral, or popliteal artery causes a critical degree of ischaemia of the leg. Injury to the superficial femoral artery and its subsequent ligation is attended by a less dangerous reduction in blood flow to the foot but the subsequent interference with function may still be considerable. In World War II De Bakey and Simeone (1945) recorded an amputation rate of 50 per cent in 2,471 arterial injuries treated at first by ligation. In 81 cases treated by suture the rate was reduced to 36 per cent. The Korean War afforded the opportunity to study this problem further and in fact Hughes (1955) was able to report an amputation rate as low as 11 per cent in cases of arterial injury treated by suture.

In peace time, arterial trauma is usually less extensive than that seen in war, and is thus more readily amenable to repair. If lateral or end-to-end suture is impossible with modern advances it is usually possible to implant an arterial graft. In most traumatic cases no serious technical difficulty exists because patients are usually healthy and the artery wall is substantially normal and holds sutures well.

Lateral injury to a main artery results in massive pulsating haematoma. Such a lesion is best approached through the haematoma after controlling the artery above and is repaired with interrupted sutures. In a recent case operated on by this technique the haematoma contained 5½ pints of blood and clot (Plate I).

Arteriovenous fistula is a late result of injury to artery and vein. Quadruple ligation and excision was the standard treatment up to ten years ago and because of the great hypertrophy of the vascular system in this condition serious ischaemia rarely resulted. However, with modern technique it is usually possible to preserve continuity of both artery and vein either by lateral suture of the fistulous opening in the artery or by end-to-end suture after excision (Fig. 1).

Restoration of the circulation is always an urgent matter whether the obstruction is due to embolus or trauma. In either case the urgency is dictated by the adequacy of the collateral vessels. When these are insufficient an important early sign is loss of cutaneous sensation. If serious ischaemia persists for more than 8–10 hours, muscle death will occur and the leg will be lost even if the circulation is eventually fully restored. In cases of embolus heparin may be used to prevent propagation of thrombosis but surgery must not be withheld if sensation is impaired. Heparin must never be used when there is an arterial injury. We have not found sympathetic block or vasodilators of any value in these cases. The only factor which improves the circulation is gravity and when the blood supply is precarious the limb should be placed a little below heart level.

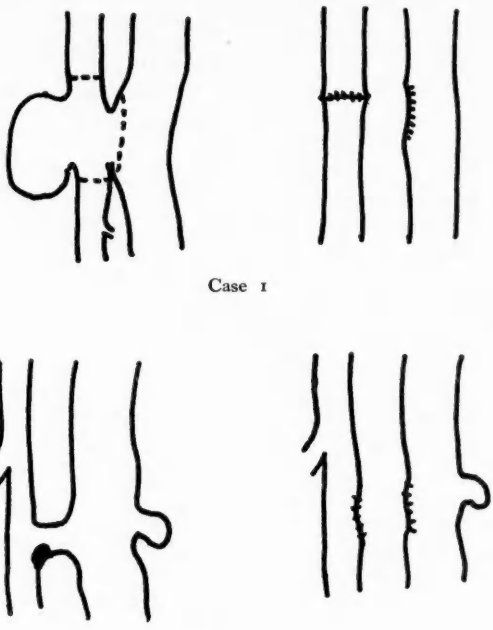
EFFECTS OF ATHEROSCLEROSIS

With an ageing population the problem of arterial narrowing due to atherosclerosis with consequent claudication or gangrene is becoming more important.

The blood flow through the narrowed artery is progressively impeded until thrombosis takes place and this extends proximally and distally from the site of obstruction to the level of branches which are wide enough to carry a good flow of blood (Fig. 2). The final length of thrombosed artery always has collateral arteries arising flush with the upper and lower limits of the thrombus.

An artery already narrowed by atheroma may thrombose while the patient is confined to bed with an illness or after an operation. In other cases thrombosis happens at night, and the patient notices his first symptoms on rising; but in the majority of cases the symptoms increase gradually and no special time of onset is noted.

When a major artery is obstructed by an embolus there is an abrupt change from a normal circulation to total arrest and symptoms are usually dramatic and severe. In atheroma the artery becomes progressively narrowed and the final episode of thrombosis does not give rise to such acute symptoms. Sometimes the thrombosis adds so little to the obstruction which is already present that it is clinically silent and the



Case 1

Case 2

FIG. 1. *Two examples of arteriovenous fistula. In case 1 the artery was repaired by end to end suture after excision of the fistula. In case 2 it was possible to do a lateral repair of the artery. The bomb fragment which caused this fistula is seen in the wall of the artery. The associated false aneurysm of artery or vein seen in both cases is a common finding.*

patient cannot give an accurate date on which it happened, but when thrombosis occurs in an artery which has only suffered a partial occlusion the effect may be considerable.

Atheromatous plaques may be generalized throughout the arterial system but in many cases they are localized to a restricted part of the circulation. A single block with good collaterals may not cause severe symptoms but a second block or severe narrowing gives rise to a much more serious state of ischaemia.

The general picture in atheroma is one of gradual circulatory deterioration. However, each incident of thrombosis is associated with some dilatation of collaterals and the natural tendency is towards some temporary improvement in many cases. In addition, subjective relief and apparent improvement results when the patient with claudication finds that he can walk considerably further if his speed is reduced. These natural phenomena must be taken into account when assessing the value of any new form of treatment.

Different Diagnosis. The only serious difficulty appears to be the distinction from sciatica or hip disease. In patients with aortic thrombosis walking may induce ischaemic pain in the buttocks; this gluteal claudication may be mis-diagnosed as pain due to osteoarthritis of the hip. Palpation of the femoral pulse clears up this difficulty immediately. On the other hand patients who have sciatica may be mis-diagnosed

as intermittent claudication. This mistake will be avoided if the peripheral pulses are felt because if they are present the patient is not suffering from vascular obstruction and it is necessary to look elsewhere for an explanation of pain in the limb.

Arteriography. In practice the only special investigation used to confirm diagnosis is arteriography, and this is only done when direct surgery is considered a possibility. Femoral arteriography is performed by percutaneous puncture; 30 ml. of 50 per cent diodrast is injected and a series of X-ray pictures exposed down the leg. Lumbar aortography is performed by a similar technique. No serious complications have so far been encountered in Bristol, though rare misadventures are well recognized. In aortography, injection of a high concentration of diodrast into the renal or mesenteric circulations may have severe consequences. When one or both femoral pulses are absent the visceral branches of the aorta are certain to receive a particularly heavy dose of diodrast and for this reason the concentration used must not exceed 50 per cent in any circumstances.

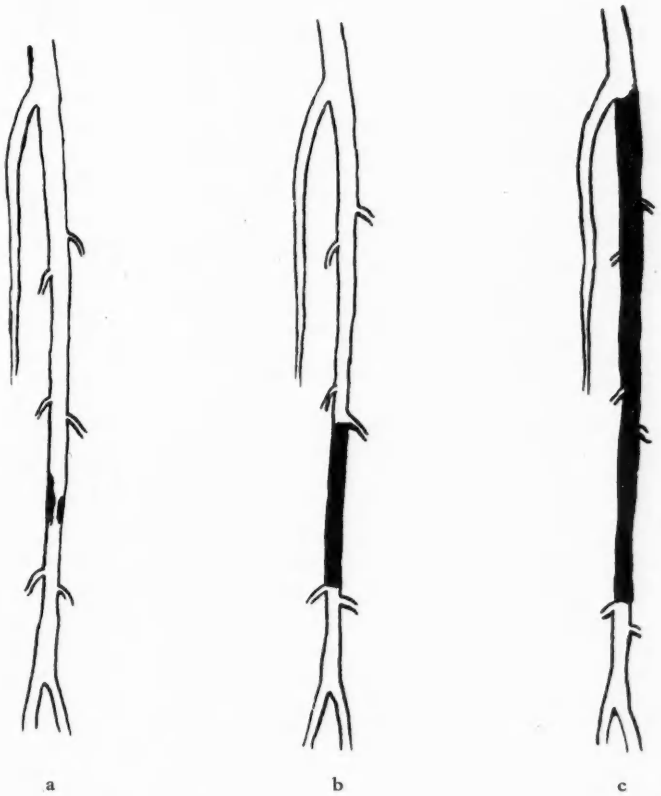


FIG. 2. (a) *Narrowing of the femoral artery by atheroma.*
(b) *Secondary thrombosis.*
(c) *Extension of thrombosis to the origin of the profunda femoris artery.*

Mis-Use of Vasodilator Drugs. Many patients attending the Out-patient Department for consultation about peripheral vascular problems are already taking vasodilator drugs. Though subjective remission is often reported there seems no objective evidence that any benefit results. In fact the use of a vasodilator drug by the oral route causes a generalized vasodilation and some lowering of blood pressure results. An additional strain is placed on the heart which is likely to be affected by atheroma and heart failure or fibrillation results in some cases. At the same time the fall in systemic blood pressure is likely to be associated with a reduced blood flow to the limb with a main artery obstruction. Blood flow studies show this to be the case. This criticism applies to all vasodilators used by the oral route. When the intra-arterial route is used the drug passes rapidly through the capillary bed and the effect s again generalized. On the evidence it appears certain that vasodilator drugs can do nothing but harm and that any subjective improvement must be attributed to the natural course of the disease.

Treatment of Arterial Obstruction due to Atheroma. The only form of treatment that provides relief in intermittent claudication is the restoration of circulation by an arterial graft. The only exception to this is that the operation of endarterectomy is usually successful when applied to short lengths of the aorta or iliac arteries. When performed on the femoral artery it is followed by early thrombosis. Lumbar sympathectomy does not improve the blood flow to the muscles though in cases presenting with early necrosis a slight increase in skin flow may sometimes be achieved and this may be sufficient to save the leg or to relieve rest pain.

Intermittent Claudication. In many cases this sympton is a nuisance but does not seriously interfere with a patient's life. In such a case an arterial graft cannot be justified, but when the incapacity is severe and the patient is unable to work or get to work, grafting must be considered. In a previous study (Horton 1956) it was shown that when grafting was performed for obstructions distal to the inguinal ligament there was an immediate thrombosis of the graft in about 50 per cent of cases and in addition there was a high incidence of delayed thrombosis due to progressive disease beyond the limits of the graft during the ensuing 6–12 months. Grafts to the iliac arteries were more satisfactory and showed a much smaller incidence of thrombosis in the same follow up period. Because of these results, grafts beyond the inguinal ligament were rarely performed in 1956 and 1957.

Recently there has been a tendency both in the United States and in this country to advise the use of much longer femoral grafts to bypass most of the femoral artery and the proximal part of the popliteal artery. At the same time the end-to-end grafting technique has been abandoned in favour of a by-pass type of operation. This operation is greatly to be preferred because immediate thrombosis is most unusual and in addition it is not necessary to sacrifice any collateral arteries in putting the graft into position. (Fig. 3). It is therefore to be expected that with the by-pass technique and using grafts up to 14 in. long, much better immediate and long term results will be obtained. Femoral grafts are therefore being performed again more readily for intermittent claudication and the early results are very encouraging (Fig. 4).

Gangrene. When gangrene is advanced the arterial disease is usually severe and grafting is impossible. In a few cases a graft may permit a minor amputation but most patients require an amputation above the knee. The patient with minor necrosis of a toe associated with severe rest pain may also require a major amputation. However, some satisfactory results of grafting are seen in this type of case and if it is technically possible, a graft should be advised because restoration of the circulation will save the leg.

Not all patients in clinical need of treatment by grafting are anatomically suitable. This can only be determined by arteriography which must show that beyond the block there is a patent artery of sufficient size to take the graft. The distal third of the popliteal artery is often suitable for attachment of a graft when the more proximal artery is very diseased. In addition there must be sufficient arterial bed in the leg to

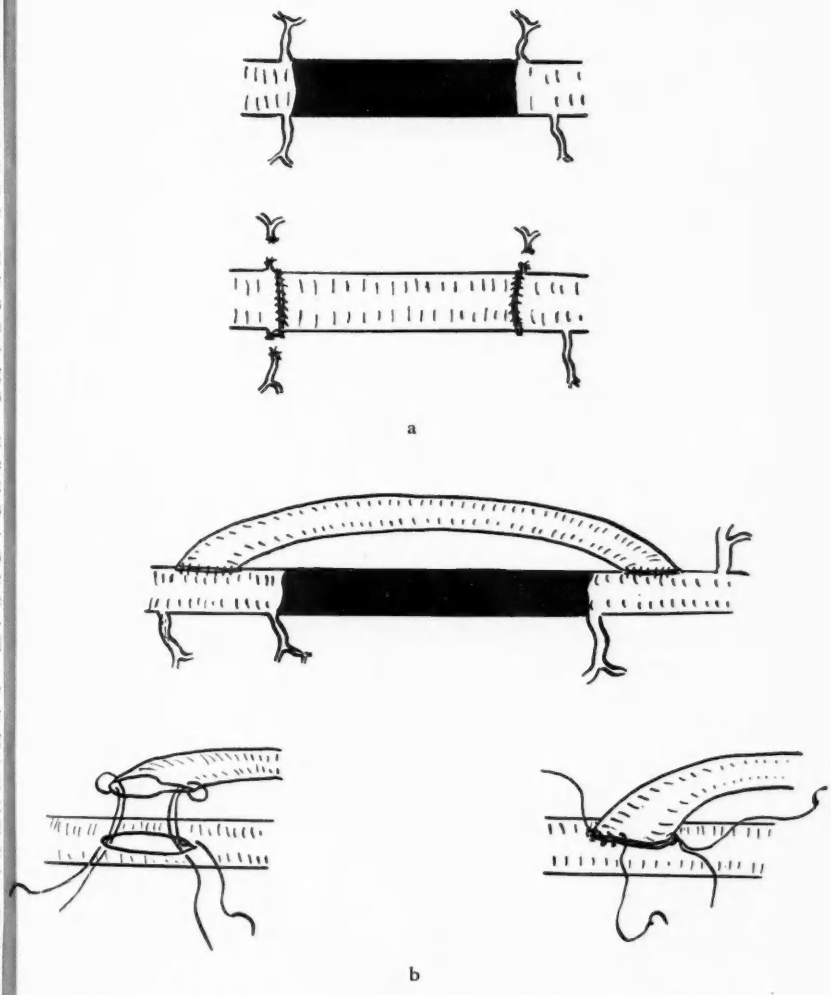


FIG. 3. (a) *This end-to-end operation is now obsolete. Note sacrifice of collaterals.*
(b) *Short by-pass operation. Collaterals are not ligated.*

take an adequate flow; at least one of the three named arteries below the knee must be freely patent.

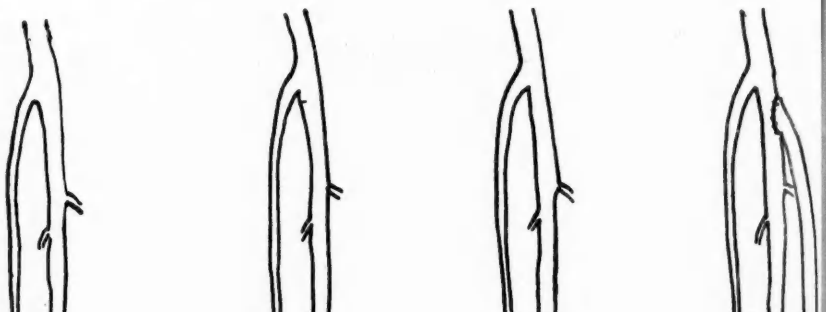


FIG. 4. *Evolution of grafting technique*

(a) *Obsolete end to end graft after excision of thrombosed segment.*
(b) *Short by-pass segment.*
(c) *Long by-pass graft.*

SUMMARY

The management of cases of arterial obstruction requires individual appraisement of every case. Those clinically requiring surgical intervention are investigated by arteriography and, if careful selection is made on clinical and radiographic evidence, very good results may be expected.

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PERINATAL MORTALITY

BY

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Sophisticated peoples have a low fertility and great concern for the welfare of their children. Their low birth rate is due to deliberate family limitation; besides planned parenthood, their desire for better living standards leads to emancipation of women, and later marriages. A century ago in Britain, when conditions of infant life were appalling, no less than 15 out of every 100 live born infants did not live to see their first birthday: at the same time, each year every 100 women of child-bearing age were producing 15 babies. Now, 97 per cent of infants survive their first year, but our potential mothers give birth at the annual rate of only 7.3 per cent.

The immense gain in infant survival has compensated for the losses due to lowered fertility. However, the point has now almost been reached where no further decreases in infant and child mortality are possible, and even if the present low mortalities were reduced to zero the increase in population would be negligible. If a fall in population is to be avoided it is necessary either to reduce reproductive wastage at an earlier stage or to create conditions favourable for increased fertility. The ideal population would combine a low infant death rate with high fertility, which is most nearly achieved by the U.S.A. where the population is increasing at the rate of 2 per cent, per annum. Although estimates of reproductive wastage are imprecise it is probable that there are each year in Britain some 250,000 miscarriages; many of these are abnormal foetuses, others are unwanted pregnancies but apart from them a great number of potential lives are lost. Once the foetus has reached viability (at twenty-eight weeks) its fate is more accurately known; the hazards of late pregnancy and delivery account for 26,000 deaths each year, a casualty list twice that of carcinoma of the lung and five times that due to fatal road accidents.

Perinatal Mortality Rate:

Death of the infant at about the time of birth is described as "perinatal". In Britain perinatal mortality is defined as "the combination of stillbirths and first-week neonatal deaths". If these are expressed as deaths per 1,000 total births the "Perinatal Mortality Rate" (P.M.R.) is obtained. This P.M.R. is a useful concept because it emphasises the episodic nature of birth in the life of the individual from conception to dissolution, and it eliminates the often arbitrary distinction between stillbirth and early neonatal death. However, the British definition of P.M.R. is neither recognised nor used widely. One objection is that lethal congenital malformations, some intra-uterine deaths and some post-natal deaths are not directly associated with the process of birth. Moreover, it is often inconvenient to present statistics sharply drawn at the seventh post-natal day.

In Britain the P.M.R. is about thirty-seven per 1,000 total births. Twenty-three of these are stillbirths and fourteen are early neonatal deaths. Unlike the post-natal death rate (i.e. deaths occurring between the fourth week and the end of the first year of life) the P.M.R. has not greatly altered in recent years, and for ten years the stillbirth rate has remained the same. Efforts to improve these figures are not a mere academic task, for this loss of life is ill-spared by the community and it also represents much personal disappointment and family tragedy. It is not always true to say that further pregnancies can make good an unsuccessful one, for too often it is the precious child that is lost, or subsequent fertility is impaired through physical causes, or by suppression of maternal feelings and desires.

The causes of perinatal mortality are diverse. Problems of maternal, placental and foetal pathology are inextricably woven into the fabric of genetic, economic and social factors. Not all pregnant women avail themselves of the present obstetrical services and skill and there are varying standards of medical care. All aspects of the problem must be investigated if it is to be solved. Research in perinatal mortality has followed three main paths. (1) Analysis and correlation of the data obtained from death certificates. (2) Clinico-pathological studies of individual cases has yielded fresh facts about pathological conditions causing perinatal death and their prevention and treatment. (3) the study of individual factors implicated in perinatal mortality have helped to find the most satisfactory methods of obstetric and paediatric care.

Statistical Approach:

The statistical analysis of the notifications of stillbirths and neonatal deaths has been most rewarding in defining the extent of the problem and the areas which are succeeding or failing to deal with it. Important correlations have also been made with other statistical data mainly of a socio-economic kind. Thus perinatal deaths are not uniformly distributed either between or within nations. For instance, the perinatal mortality of Wigan is twice that of Oxford, while Bristol comes close to the national average[1]. Interpretation of this statistical information is of course limited by the uncertainty of the data provided by birth notifications and death certificates. If mortality data are to be compared on an international scale, both terminology and presentation of facts must become uniform. At present this is not so, but the deliberations of the Statistical Commission of the United Nations and the W.H.O. Committee on Health Statistics should secure common agreement on these matters. It is not only statistics on the grand scale which make valuable contributions. Such relatively modest surveys as those carried out in Bristol[2] and the S.W. Region[3] have produced results of more than local interest. Both these surveys showed that in an economically-thriving population there were no significant differences in perinatal mortality between one social class and another. In neither of these surveys could poor medical care be held responsible for more than a small proportion of deaths. In the Bristol series, 8 per cent of deaths might have been prevented by better co-operation of the patient, by more competent obstetric management or by more adequate services. On the other hand thirty-five per cent of perinatal deaths in New York City[4] were recently held preventable on the grounds of inadequate prenatal care, error in medical judgment, error in medical technique, family at fault, unqualified medical attendant or inadequate paediatric care. A direct comparison between these two observations is possibly not valid, but we must not be complacent about our own mortality figures. Whatever the true position it is important that our maternity services should be based on knowledge rather than prejudice, whimsy and politics.

Clinico-Pathological Study:

While statistical information is vital for the demographer and the administrator it is anathema to many clinicians. They are concerned with the management of individual pregnancies and not the manipulation of masses of gravid units. The clinical obstetrician and paediatrician have much to learn from the detailed clinico-pathological study of many individual infant deaths. Such work can only take place in maternity departments which deal with large numbers of cases and command expert clinical and pathological services. Maternity units of this quality usually admit cases selected on medical or social grounds and thus tend to deal with a high proportion of abnormal midwifery. Mortality figures from such a source do not reflect the over-all problem of perinatal mortality, but they do indicate clearly the types of lesion encountered. The most detailed recent study of perinatal mortality undertaken in Britain is that of Bound, Butler and Spector[5] from University College Hospital. They divided deaths into three main groups: antepartum, intrapartum and postpartum deaths. Antepartum deaths were further differentiated into those, without evidence of

asphyxia (usually macerated) and fresh stillbirths with signs of asphyxia. The former were most often due to maternal toxaemia, high maternal age, small antepartum haemorrhages, maternal diabetes or rhesus incompatibility. Acute asphyxial deaths had similar maternal histories, but catastrophes such as severe antepartum haemorrhage or prolapse of the cord were also frequently found. Intrapartum deaths were anoxic or traumatic in origin and were associated with prolonged or complicated labours. Postpartum deaths occur against an overwhelming background of prematurity, although birth trauma and infection are not uncommon.

For several years perinatal mortality has been closely studied at Southmead Hospital, Bristol. Dr. Norman J. Brown[6] has kindly given me permission to publish the results of his careful post-mortem investigations on perinatal deaths. In the three years 1955–57, there have been 739 stillbirths and neonatal deaths at Southmead Hospital, almost all of which fall within the category of Perinatal deaths. Of these, 59 per cent were stillbirths, 26 per cent neonatal deaths in premature infants and 15 per cent neonatal deaths in full-term infants. In 648 deaths (88 per cent) the cause was accurately determined on a basis of clinical and pathological studies. Lethal congenital malformations (the majority being anencephaly or spina bifida) were responsible for 164 deaths (22 per cent). While it is not intended to minimise the importance of congenital malformations as a cause of reproductive failure, they are not strictly relevant to the present discussion. Whether congenital malformations are due to genetic or environmental causes—or the facilitation of one by the other, it is generally accepted that with rare exceptions they are determined during the first three months of pregnancy and are thus not part of the hazards of late pregnancy. If these deaths are excluded as not resulting primarily from perinatal causes there remain 494 autopsies the findings of which are available. Excluding lethal congenital malformations altogether, we found 270 stillbirths (55 per cent) 153 premature neonatal deaths (33 per cent) and 53 full-term deaths (12 per cent). Pre-eclamptic toxaemia was the preeminent cause of perinatal death. Maternal toxaemia or hypertension was associated with 20 per cent of the stillbirths and was responsible for the early onset, or induction of labour in 21 per cent of the premature deaths. Other major causes of stillbirths were ante-partum haemorrhage and placental infarction not due to toxaemia. Birth trauma and acute intrapartum asphyxia was responsible for many fresh stillbirths. Postmaturity, maternal diabetes and erythroblastosis also appeared as causes of foetal death.

Effects of Prematurity:

Consideration of the neonatal deaths show how great is the problem of prematurity. While only 6 or 7 per cent of viable pregnancies terminate prematurely, these premature deliveries account for 74 per cent of all neonatal deaths. Most of the deaths (77) in premature infants are associated with pulmonary inadequacy. At autopsy, atelectasis or gross immaturity of the lungs is found. Intra-ventricular cerebral haemorrhage, probably asphyxial in nature, is frequently associated with pulmonary atelectasis; in a few cases intra-cranial haemorrhage is obviously traumatic in origin. The clinical aspects of these premature deaths are perplexing. Pulmonary inadequacy is usually manifest not at birth but a few hours after when tachypnoea and grunting respirations appear; the condition worsens with retractions of the ribs or sternum, intense dyspnoea, cyanosis and apnoeic attacks; often the infant becomes, and remains, oedematous. At autopsy so-called "hyaline membranes" are frequently found distributed around the walls of the alveolar ducts which are surrounded by atelectatic alveoli. Apart from a consistent relationship with prematurity, infants of diabetic mothers and rarely full-term infants born by Caesarean section, no fundamental facts are known about the aetiology of this pulmonary syndrome. Because treatment of this condition is ineffectual and the proper management of prematurity is exceedingly expensive, prevention of prematurity is far more important than its cure.

Few live-born full-term infants without serious congenital malformations die.

Trauma, infection and erythroblastosis are the conditions most likely to cause death. Birth trauma might be prevented by alternative methods of delivery, infection is now an inseparable hazard of hospital midwifery, and erythroblastosis should cause very few neonatal deaths were ante-natal prediction and well-timed delivery always practised[7].

Study of Special Aetiological Factors:

The third investigational approach to perinatal mortality is the study of all the factors concerned or believed to be concerned in diminishing the infant's chance of survival. In a short review it is impossible to survey all the reports which are relevant to the concatenation of events which may lead to a dead baby. Amongst these must be included any deviation from the normal in the anatomy or physiology of the mother; the adequacy of the placenta and the integrity of the umbilical pathway; the capacity of the pelvis and the forces of labour; the size and maturity of the foetus, its presentation or organisation, all influence the outcome of pregnancy. Interference with natural parturition by drugs or instruments may do great good but, used unwisely, may be harmful. The majority of these factors have been analysed and we believe that modern obstetric and paediatric practice is based on scientific facts; however, much has yet to be learned and many of our traditional procedures are over-due for critical review.

Probably the largest single factor contributing to perinatal mortality is pre-eclamptic toxaemia. Its aetiology still remains a mystery in spite of recent research. Much of its baneful effects on the foetus can be minimised by good ante-natal care; this has been clearly shown by Hughes[8] who has by empirical measures greatly reduced the incidence of severe toxaemia and cut down the premature neonatal death rate. The recent work of Robinson[9] suggests that salt added to the diet of the pregnant woman not only prevents toxaemia but may reduce by almost half the perinatal death rate; if this is confirmed it will revolutionise our treatment.

Another hotly debated factor in perinatal death is postmaturity. All the pontifications of the pundits indicate that the practitioner need not be unduly alarmed. Provided that elderly primiparae and women with the slightest toxaemia or hypertension are not allowed to progress beyond term it seems that interference is probably unjustified.

The management of prematurity is also undergoing revision. When should the umbilical cord be clamped? We were told some years ago to delay clamping it until pulsations ceased because the premature baby needed the extra blood from the placenta to prevent subsequent anaemia. Later we were then told that the baby had too many red cells and that by delayed clamping we were contributing to neonatal jaundice. Now we no longer believe that neonatal jaundice is due to excess haemolysis (apart from blood incompatibility) and once more we must let the placental blood drain into the baby lest there be inadequate blood volume to erect the lungs[10]. Heat, humidity and oxygen were once the major therapeutic weapons for all the ills of the premature infant—for did not babies die of anoxia and did they not emerge from a hot, wet environment? The babies which survived this enthusiastic treatment with oxygen often developed retrolental fibroplasia and became blind. To-day it is reasonably suggested that premature infants should be nursed in artificially low concentrations of oxygen[11] and that hypothermia is beneficial. The last word has not been written on any aspect of pregnancy, parturition or paediatric care and only by continuing research will all the truth be known.

The latest method of acquiring knowledge about perinatal mortality is the large-scale socio-medical enquiry. This technique, derived from the discipline of Social Medicine, should prove useful in the field of perinatal mortality. For one week in March this year a detailed questionnaire was completed on every birth in Britain and covered almost every relevant social and medical factor. As many perinatal deaths as possible were referred to selected pathologists for detailed examination and the results

will be correlated with the obstetric and paediatric histories. This survey is being carried out under the auspices of the National Birthday Trust; it is hoped the facts so found will show those areas where maternity services might be improved and will indicate the fields where intensive research is needed. The survey comes at an opportune time, not only because of the present interest in perinatal death but also because a revision of maternity services is now being considered. This revision may well affect the professional lives of many family doctors and may alter the whole structure of the profession of midwifery.

I wish to acknowledge the help given by the Obstetricians and Paediatricians at Southmead Hospital, Bristol and especially the kindness of Dr. N. J. Brown who has made available pathological material and reports.

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AN EXHIBITION OF IMPORTANT ANATOMICAL BOOKS

A. E. S. ROBERTS

Medical Library, University of Bristol

"It is not necessary for the understanding of any science that we should be acquainted with its history", said Matthew Baillie in 1785. "There is, however", he went on to say, "a strong curiosity prompting us to inquire into the history of discoveries in any branch of science. . ."

This quotation comes from *Lectures and Observations on Medicine* by the late Matthew Baillie, printed in 1825 for private distribution among his friends according to directions left in a codicil to his will. The first part of his book consists of two introductory lectures to the course on anatomy given in Great Windmill Street in which he gives a very good outline history of anatomy.

In the Medical Library of the University of Bristol there are many books by authors who contributed something to the development of human anatomy, and a few of the important ones are at present on display in the Library.

Herophilus (300 B.C.) is said by Galen to be the first person publicly to dissect both human and animal bodies, and although his works are lost, Galen tells us that he recognized the brain as the central organ of the nervous system. We owe to him the first description of the lacteals. He was the first to grasp the nature of nerves other than those of the special senses. He divided nerves into motor and sensory, but continued to use the word neuron also for sinews and ligaments. He described the meninges and the torcular Herophili, which is named after him. He distinguished the cerebrum and the cerebellum, the fourth ventricle and, above all, the calamus scriptorius, which he named. The terms prostate and duodenum are derived from those which he used. He made the first clear distinction between arteries and veins.

Erasistratus, a younger contemporary of Herophilus, extended the observations of Herophilus on the lacteals. He also regarded the heart as the source of both arteries and veins and came very near to discovering the circulation of the blood. He realized the existence of the capillary system.

Galen (A.D. 130–200), in his voluminous works, gathered together all the medical knowledge of his time. His own knowledge of anatomy is best seen in his *De Anatomicis Administrationibus*, a translation of which is on display. His works dominated medicine until the time of Vesalius, thirteen centuries later.

It was not until the fourteenth century that dissections were generally legalised, so that in the time of Leonardo da Vinci (1452–1519) dissection of the human body was a well-recognized feature of medical instruction. Yet those early Renaissance anatomists made no progress, in spite of their opportunities for direct observation, for the reason that the lecturer not only remained aloof from the cadaver, but slavishly followed the written text; the actual dissector was inefficient and the process was necessarily a hurried one since there was no means of preserving the bodies from putrefaction.

The *Fasciculus medicinae* compiled by Johannes de Ketham in 1491 is the first printed medical book and consists of a collection of short medical treatises. In the library there is a facsimile of the 1493 edition which contains the *Anathoma* by Mundinus, the first book devoted solely to anatomy. This is on display open at a page illustrating an anatomy lecture. After the invention of printing the *Anathoma* was issued in several editions so that, having been originally written in 1316, it was in use altogether for well over 200 years.

Leonardo da Vinci in his anatomical drawings represented the structures of the human body with an accuracy and skill such as had never been seen before. A comparison of the typical fifteenth century *situs* figure in the *Fasciculus medicinae*

with Leonardo's *situs* figure (figures 3 and 4 in McCurrich, *Leonardo da Vinci, the anatomist*) shows how far Leonardo was ahead of his time in the matter of anatomical illustration.

The next book displayed is the most fully illustrated of the pre-Vasalian anatomies, *De dissectione partium corporis humani* by Charles Estienne, part of which was in preparation as early as 1530 though it was not published until 1545. The text is largely dependent on Galen. The chief defect of Estienne's work is the distorted ugliness of his figures, but they are the earliest, except for those of Leonardo, in which whole systems, venous and nervous, are shown.

The last of the pre-Vasalian illustrated anatomies is that of J. B. Canano (1515–1579), *Musculorum humani corporis picturata dissectio*, circa 1541. This pamphlet, a facsimile of which is displayed, was unique for its time in exhibiting each muscle in a separate figure and in approximately correct relation with the bones. Unfortunately the beautifully drawn figures, owing to the poor paper, have greatly deteriorated since his day.

In his superbly printed *De fabrica corporis humani*, Vesalius (1514–1564), having cast off the shackles of Galen, confined himself to what could be seen in the body. Not the least of the distinctions of his great work (a copy of the first edition 1543 is shown) is the dramatic and living impression that he succeeded in imparting to his figures which were so admirably executed by Jan Calcar, a pupil of Titian. Garrison and Morton consider the publication of this book was the greatest event in medical history since Galen: it ranks second only to Harvey's *De motu cordis* in importance.

The work of Bartolommeo Eustachius (1520–1574) *Tabulae anatomicae* was completed in 1552 but this fine collection of plates remained unprinted and forgotten in the Vatican Library till 1714 when they were published by Lancisi. Charles Singer is of the opinion that had these plates, which are more accurate than the work of Vesalius, been published in 1552, Eustachius would have ranked with Vesalius as one of the founders of modern anatomy.

A 1722 edition of his *Tabulae anatomicae* is in the Library, but it is too large to put on display. The same applies to *Tabulae anatomicae LXXIIX* (1632) by Guilio Casserio (1552–1616) and *Anatomia humani corporis* (1685) by Govert Bidloo, both of which contain magnificent copper-plate engravings.

Asellius (1581–1626) first distinguished the lacteals, at the beginning of the seventeenth century, to be a particular system of vessels, yet he was mistaken about their termination: for he believed that they ended in the liver, the trunk of the absorbents being still unknown. Attention to this subject was now, however, awakened among anatomists, and the error of Asellius was soon corrected by Pecquet (1622–1674). It remained still, however, to make the same progress in the human subject and Veslingius (1598–1649) is said to have observed in 1634 the lacteal vessels in man, and in 1649 the thoracic duct, and the lacteals were soon traced to their termination in it. The lacteals, however, form only a small part of the absorbent system and Rudbeck (1630–1702) has the honour of discovering the lymphatic vessels in 1650 while still a student at Padua, but he did not make the discovery public till 1653, a few months after Bartholin. Although the lymphatic vessels were now certainly discovered, yet anatomists were entirely ignorant of their use, and their relation to the absorbent system. This further important discovery was reserved for William Hewson (1739–1774).

The following books illustrate the history of the absorbent system: Gaspare Aselli, *De lactibus sive lacteis venis, in* Spigelius—Opera omnia, 1645 (too large to display). Aselli's original work, published in 1627 includes the first anatomical plates printed in colours, but the library unfortunately does not possess this. The works of Pecquet, Bartholin and Rudbeck, are contained in a collection by S. Hemsterhuys entitled *Messis aurea exhibens: anatomica: novissima et utilissima experimenta* 1659. This little book is on display.

William Hewson's *Experimental inquiries. Part II. Containing a description of the*

lymphatic system . . . 1774 is on display and the library also has the other two parts of his "Experimental inquiries" making in its complete form a rare and valuable possession.

The seventeenth century, owing to the invention of the microscope, produced some brilliant microscopical investigators; also the discovery of the process of making permanent anatomical preparations by means of injections opened up a new field of interest among anatomists. The study of anatomy had reached such proportions that it was now necessary to consider the preparation of the object under examination and following that the art of injection. But for injection to be satisfactory it was necessary to discover both the most suitable material and the means of adequately making the injection. We have Swammerdam (1637–1680) to thank for discovering the most suitable material and De Graaf (1641–1673) for inventing the syringe. A copy of De Graaf's *Opera omnia*, 1677, is on display, open at the illustration of his syringe which accompanies his work *De usu siphonis in anatomia.*

Marcello Malpighi (1628–1694) was one of the most brilliant microscopists of the seventeenth century. His most important discovery was capillary circulation which he described in 1661 in the lungs of a frog. It was announced in *De pulmonibus observationibus anatomicae* which consisted of two letters to Borelli, the second of which contains his discovery. It is republished in his *Opera omnia*, Vol. 2, p. 331, (which is on display), and an English translation by J. Young appeared in Proceedings of the Royal Society of Medicine, 1929–30, Section of the History of Medicine, vol. 23, pp. 1–11.

The anatomists of this period took immense pride in their preparations and paid more attention to their beauty than to their instructiveness. Paramount in this respect was Frederick Ruysch (1638–1731) a volume of whose *Thesaurus anatomicus* is exhibited. His name is connected anatomically with several discoveries, among which is a description of the valves of the lymphatics. The exact composition of the material used for injection by Ruysch, and also by many other microscopists, was kept secret.

The seventeenth and eighteenth centuries abound in originators of eponymous anatomical terms, and it is impossible in a short article to name all the anatomists who are perpetuated in medical terminology. Nor is there room to display all the relevant books possessed by the library. Among them, however, mention might be made of Spigelius (1578–1624) who has given his name to the Spigelian lobe of the liver; Francis Glisson (1597–1677) who was the first to give an accurate description of the liver and described the so-called Glisson's capsule; Thomas Willis (1622–1675) who in his *Cerebre anatome* gave the most exact account of the nervous system that had yet appeared. The Circle of Willis is still called after him.

Other names which might be mentioned are Stephen Blankaart (1650–1702) who succeeded for the first time in 1675 in injecting the capillary vessels, by which he obtained permanent and objective proof of the connection between the arteries and the veins; Giovanni Battista Morgagni (1682–1771) who founded modern morbid anatomy by his work *De sedibus et causis morborum*, in which he attempted to bring the symptoms of disease observed during life into relation with the phenomena revealed at autopsy; Jean Baptiste Senac (1693–1770) who wrote a valuable work on the structure of the heart which is said to have laid the foundation of cardiac pathology; William Cheselden (1688–1752) applied himself especially to osteology as did James Douglas (1675–1742); the elder Alexander Monro of Edinburgh founded the Medical School there; Albrecht von Haller (1708–1777) who was a prolific writer on anatomical subjects and whose textbook of physiology was used for many years; he was also an authority on the history of medicine; and John Hunter (1728–1793) who was one of the greatest surgical investigators the world had seen and who was eminent in every department he touched.

During this time anatomy consisted of a collection of descriptions of the different organs and groups of organs. But Xavier Bichat (1771–1804) in his *Anatomie générale appliqué à la physiologie et à la médecine* called general anatomy into existence when

he distinguished the tissues common to all or peculiar to each part of the body. He thus changed the whole outlook of anatomy and physiology. His *Anatomie générale* is one of medicine's most important books.

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HEREDITARY TELANGIECTASIA: HODGKIN'S DISEASE

(A Clinical Pathological Conference of the University of Bristol Medical School)

CHAIRMAN: PROFESSOR T. F. HEWER

Dr. Cates: This man, who was employed as a fireman in an aircraft factory, died from vomiting blood on the 7th December last year. He was a known victim of familial telangiectasia and had had several previous episodes of haemorrhage from the gut which had been very severe. Before he died he developed a fresh disease for which he received a course of nitrogen mustard.

In 1930, at the age of 31, he had a severe nose bleed, losing about one pint. He went to the General Hospital where his condition was recognized and he attended there on and off for the next twenty years, with bleeding occurring up to twenty times a year.

In 1932 he developed red spots on his face, nose and lips and then began to bleed from his tongue which was cauterized, and this stopped the bleeding. He was given a course of deep X-ray to the spleen (a treatment that would not nowadays be used for this condition). Nose bleeds continued for the next twenty years, otherwise he led a normal life.

In 1952 his haemorrhages became worse and he was subjected to a course of oestrogen therapy. It had been noticed that women with telangiectasia bleed more readily in the second two weeks of the menstrual cycle when oestrogen is not in the ascendant but progesterone is, so they thought it would be worth keeping him permanently under the effect of oestrogen. The effect on him of the intensive course was that he became impotent, his breasts became sore and swollen, he said he felt "awful" and that his nose was no better but bled just as much.

About 1956 he was referred to Mr. Pocock because he was having melaena. This was the first haemorrhage from the gut, the previous bleeds were all from the nose and mouth. As he also had indigestion the problem was whether this was bleeding from telangiectasia in his gut or had he two diseases. Did dyspepsia indicate an ulcer?

He was admitted and underwent gastroscopy. Mr. Capper reported that to every square inch of stomach lining there were three telangiectases and since there was no ulcer the bleeding must come from them. He was treated with iron and blood transfusion and was readmitted with a severe haemorrhage of the same sort in January and June 1957. He was also admitted to Frenchay Hospital as an emergency in the summer of 1957 with a severe nose bleed again.

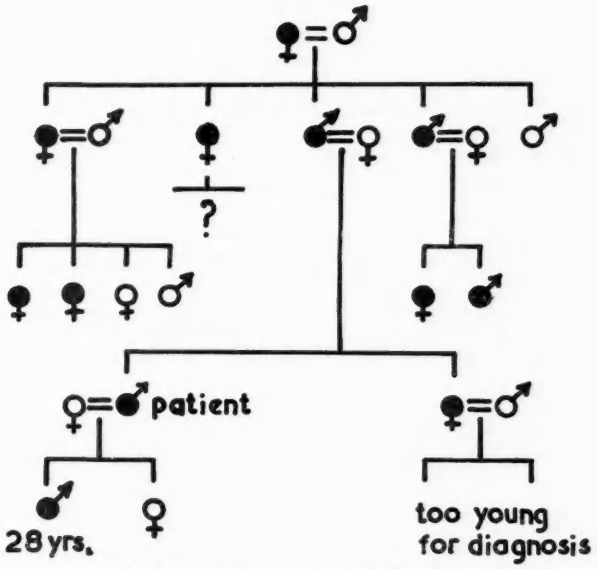
About this time we wondered whether he should be subjected to a rather heroic form of surgery in which, Mr. Capper says, the stomach can be opened up and bleeding points cauterized; but before this was decided something else happened—though I'll come back to that later.

In December he had a profuse haemorrhage and was readmitted as an emergency and had altogether 13 pints of blood. At this time he had haematological changes which may be related to the treatment I am going to describe in a minute. During haemorrhages he became severely shocked and blood pressure went right down and stayed down. There was difficulty in matching blood because of his second disease and before he died his non-protein nitrogen rose to high levels and we thought his kidneys might be suffering as the result of an incompatible blood transfusion or of shock between transfusions. We decided that we were overtreating a man with another disease and put him on heroin and morphia; he died five days after admission.

Now, returning to the second disease, in September last year he had had 'flu, or what the layman calls 'flu, i.e. fever of unknown origin, headache, sweating, pains in

limbs and weakness. In October he noticed swelling in the left side of the neck followed by swelling in his axillae. He was seen by Dr. A. D. M. Smith in the Outpatient Clinic where he was found to have a high temperature, and a large liver and spleen. It seemed likely that he had an acute reticulosarcoma. A biopsy was carried out and in view of the histological changes, which Dr. Johnson will describe later, it was decided to treat with nitrogen mustard. Two injections of 6 mgm of nitrogen mustard were given intravenously (the full course is six injections of 6 mgm) and his response was dramatic. His temperature dropped to normal and stayed normal, the patient himself felt miraculously better after the injections and the lumps themselves melted away completely after these injections. At this time it was noticed that he had a few petechiae and we were watching him for haematological changes as a result of this therapy. His W.B.C. was 2,000, platelets 40,000, at which level it was thought wisest to give no further treatment. Nitrogen mustard is known to be a dangerous substance, particularly from a haematological point of view, but when you have a patient critically ill with a malignant disease like this, the opinion is that it is better to make him feel better for the time being. His platelets went down to 40,000 and that perhaps contributed to the difficulty in controlling his haemorrhage when he came in a month later and died from the bleeding.

DIAGRAM I



Family tree of the patient under discussion.
Those affected are shown in black

The family history: This condition is transmitted as a Mendelian dominant. You will see from the chart (Diagram I) that one of his children, aged 28, also has telangiectasia. The patient inherited it from his father, and three of the father's brothers and sisters had it; in fact only one of them did not. Of his cousins three out of four in one family and both members in another family had it. There are other descendants from the patient's sister who are still young, and, as you probably know, the condition

is not clinically obvious until the age of 35 to 45 or upwards, which makes it very difficult to prevent the transmission of the disease by eugenics.

Dr. Johnson: The histological appearance of the gland removed at biopsy showed complete disorganization of architecture with loss of lymph follicles and cellular invasion of the capsule. There was an increase in reticulin throughout the gland but little fibrosis. Cytological examination showed replacement of the normal elements with two types of cells, giant cells and reticulum cells together with a uniform admixture of lymphocytes.

This appearance is seen when Hodgkin's disease is advancing unusually rapidly and has been given the name Hodgkin's sarcoma.

Professor Hewer: This shows a much more malignant appearance than the usual run of Hodgkin's although there is every sort of gradation between this and the classical appearance. The term "Hodgkin's sarcoma" is not a very exact one. It is just an extremely active variant of the disease.

Dr. Lloyd: Could Dr. Cates give an explanation as to why oestrogen therapy is thought to influence bleeding from the telangiectases.

Dr. Cates: I feel this therapy is probably erroneous. The observation was that in premenopausal women epistaxis in this condition is sometimes worse in the second two weeks of the menstrual cycle, and so it was presumed that administration of oestrogen would decrease the bleeding. There are reports which indicate that oestrogens may in fact have some effect but even so I think it is clearly over-enthusiasm to treat a man to the point of impotence in order to reduce his nose bleeds; and when you give oestrogens to women in large doses there is the added danger of causing malignancy somewhere. Another patient who has had oestrogen for the same condition recently had her uterus out for carcinoma of the body.

Dr. McConnell: If oestrogens have anything to do with it would you not expect it to be a less dangerous condition in women than in men?

Dr. Cates: I don't know.

Professor Hewer: Incidentally a little impotence might be a good thing.

Professor Neale: Could you explain why there was splenomegaly 25 years before Hodgkin's disease was diagnosed?

Dr. Cates: I don't know if that is so. I presume that at that time he had an iron deficiency anaemia from bleeds and, as occurs in 30 to 40 per cent of cases of that sort of anaemia, he had splenomegaly.

Dr. McConnell: Would Dr. Lloyd tell us if extra-uterine bleeding was affected by the oestrogens? If effective such treatment would be invaluable where transfusion is not available. Is there any evidence that oestrogens are effective in dealing with extra-uterine haemorrhages?

Dr. Lloyd: I don't know.

Dr. Johnson: Dr. Harrison reported that he got successful results in four patients with familial telangiectasia and when he stopped giving oestrogens the bleeding recurred. One interesting point is that biopsies were taken from the nasal mucosa before and after they received oestrogens and the only change noticed was that the normal epithelium had turned into a squamous one. A suggestion was made that it was because of this change in the eipthelium that the nasal telangiectases were less likely to bleed.

Dr. Tovey: Three patients at Southmead have been treated by oestrogens but without success and nothing has been published.

Professor Neale: This seems to be a case of history repeating itself, twenty to twenty-five years ago uterine extracts were recommended for haemophilia.

Dr. Cates: The trouble is that negative findings are usually left unsung; if this work were published it would save other people from duplicating the experiments to get the same negative result.

Professor Neale: Do we know the age range of the members of this family who are not affected?

Dr. Cates: No.

Professor Neal: Was there any evidence of retinal telangiectasia?

Dr. Cates: I don't know.

Professor Neale: Isn't this a disease in which no retinal changes are found?

Dr. Cates: Yes.

Dr. Spence: If he had not developed disease No. 2 what was his likelihood of living a normal life?

Dr. Cates: These people do die of haemorrhage when they have developed about as widespread telangiectases as he had, they have big haemorrhages needing blood transfusions one after another.

Dr. Tovey: The oldest I know is a woman of sixty-five who has now reached the stage of transfusions every fortnight.

Dr. Johnson: At post-mortem this man had about a dozen telangiectases palely outlined on the lips. Straw coloured fluid was present in the abdominal cavity, the peritoneal surface was slightly blood-stained and there were blood-stained effusions in the pleural cavities. The lungs showed a little patchy polymorph infiltration. The chambers of the heart were normal but on the cusps of the mitral valve there were some so-called marantic vegetations (Interruption: Not endometrial tissues from the oestrogens?), made up of organizing fibrin, an incidental finding of little importance.

The condition of telangiectasia was first described by Babbington in 1865 (not by Rendu in 1868 or Osler in 1904 although given the name Rendu-Osler disease!) (Laughter).

One family tree described in the literature particularly beats this case; it is that of a Mormon in Utah who had four wives and twenty-two children, of whom ten were affected and up to a few years ago amongst the descendants who had been traced eighty-two had been affected.

Telangiectases were demonstrated in the internal organs, best seen in the capsule of the liver. Microscopically the lesion consists of a network of dilated capillaries lined only by a single layer of endothelium and dilated venules containing a little elastic in their wall. It is, in fact, a capillary-venous haemangioma. You will note that it is different from the spider naevus in which a little tortuous arteriole rises in the centre to form a pulsating body from which small dilated vessels, the legs, run out.

The stomach was examined to try and find the exact source of the haemorrhage but unfortunately this man had been dead some time and the mucosa was so heavily blood-stained it was impossible to distinguish any telangiectases. A section of stomach wall taken at random, however, showed numerous dilated capillaries.

The changes produced in the appearance of the lymph nodes by the two recent doses of nitrogen mustard were quite striking. At post-mortem none of the lymph nodes was more than about twice normal size. When first cut the surface presented a translucent gelatinous appearance from oedema but when fixed it was white and opaque. Compared with the biopsy the most striking change is an almost complete disappearance of the reticulum cells, their place being taken by small adult lymphocytes. The architecture of the lymph node is just as abnormal, however; the reticulin has become condensed, there is infiltration of the capsule and one can still make out abnormal giant cells, the remnant left behind after the disappearance of so much of the malignant reticular tissue.

Hodgkin's disease is one of the few malignant reticuloses which react fairly well to irradiation or treatment with cyto-toxic drugs. The cytological changes demonstrated in this case are similar to those which would have been seen after irradiation but take a little longer to appear.

The spleen was enlarged, congested and contained a large number of lymphocytes and a few abnormal reticulum cells.

The kidneys were swollen but showed no changes other than interstitial oedema, There was no tubular necrosis.

The femur showed extension of the red marrow throughout the upper two thirds of the shaft. This is a reactive hyperplasia in response to blood loss and possibly, but we have no proof, to a haemolytic reaction to the Hodgkin's disease.

Question: There is no evidence, is there, that Hodgkin's is produced by irradiation as are the leukaemias?

Dr. Johnson: I don't think so.

Professor Hewer: Would anybody like to tell us about the cytotoxic action of nitrogen mustard?

Dr. Johnson: I don't think anyone really knows. Irradiation produces ionization in the cells, so it is said. Nitrogen mustard probably interferes with cell metabolism in some other way but the end result is the same.

Student: What is the difference between a spider naevus and a telangiectasis?

Dr. Cates: The difference at the bedside is that a spider naevus has an arteriole in the centre and a leash of dilated capillaries coming from that and if the arteriole is obliterated by pressure with a sharp instrument all the legs of the spider disappear at once. They are mainly seen on the face in ordinary people but particularly in pregnant women and in cases of cirrhosis of the liver. Telangiectases on the other hand are deep blue blotches, often slightly raised.

Dr. Johnson: The spider naevus is a much brighter red.

Dr. Barritt: How often do marantic thrombi on the mitral valve give rise to carotid embolism?

Professor Hewer: I don't know that they do.

Dr. Barritt: I have seen one case of carcinomatosis with hemiplegia from a marantic thrombus.

Professor Hewer: I am surprised. They are usually firm, tough vegetations which do not break off and form emboli.

Question: Can we conclude that the best treatment of telangiectases is to leave them alone and merely treat the bleeding as it happens?

Dr. Cates: I do not think we have yet given Mr. Capper a fair trial to open the stomach and cauterize the telangiectases.

Dr. Lloyd: Is it really justifiable to open the stomach? Surely you find the lesions in the small intestine too?

Dr. Cates: I am not sure. Sometimes you do see cases in which, when you open them up, you find some localized areas of dilated vessels.

Professor Neale: I know a doctor who has this disease, he has a very big practice and lives a very busy life. The last time I saw him he was 66. Probably it is a good thing he didn't know about X-ray therapy.

Dr. Cates: Had our patient any arterio-venous aneurysms in the lungs?

Dr. Johnson: No.

News from Societies

PROGRAMMES FOR 1958–59 SESSION

BRISTOL MEDICO-CHIRURGICAL SOCIETY

Secretary: Dr. B. E. McConnell, 4 Napier Road, Bristol 6

8th Oct.: **Annual General Meeting. Presidential Address.** H. L. Shepherd, Esq., CH.M., F.R.C.O.G.
12th. Nov.: **Clinical Meeting.** Bristol General Hospital.
10th Dec.: Professor R. W. Scarff, F.R.S.ED., **"Breast Cancer".**
14th Jan.: Leslie C. Hill, Esq., M.D., F.R.C.P., **"Pain".**
11th Feb.: Sir Walter Mercer, F.R.C.S.ED., **"Shoulder Pain".**
11th Mar.: H. Gordon Mather, Esq., M.R.C.P., **"Cardiac Iscaemia".**
8th Apr.: Kenneth G. Malcolmson, Esq., F.R.C.S., **"Rhinitis".**
13th May: T. J. Butler, F.R.C.S., and W. M. Capper, Esq., F.R.C.S., **"Aftermath of Gastrectomy".**

THE DEVON AND EXETER MEDICO-CHIRURGICAL SOCIETY

Secretary: Dr. Charles Seward, 20 West Southernhay, Exeter

16th Oct., at 3.30 p.m.: Lambert Rogers, Esq., C.B.E., V.R.D., M.D., M.SC., F.R.C.S., F.R.A.C.S., F.A.C.S., **"Some Thoughts on Surgical Progress".**
6th Nov., at 3 p.m.: **Clinical Meeting.**
28th Nov., at 8.15 p.m.: Robert Nevin, Esq., T.D., M.A., F.R.C.S., **"The Surgery of Ulcerative Colitis".**
11th Dec., at 8.15 p.m.: **Clinico-Pathological Demonstration.**
8th Jan., at 8.15 p.m.: President's Party for Members and their wives in Board Room, Royal Devon and Exeter Hospital. Film: **"A Two-year old Goes to Hospital",** in Medical Library.
29th Jan., at 8.15 p.m.: **Clinical Meeting** at P.E.O.H.
9th Feb., at 8.15 p.m.: Lewis Couper, Esq., M.B., CH.B., D.P.M., **"The Psychosomatic Approach".**
12th Mar., at 3 p.m.: **Clinical Meeting.**
9th Apr., at 8.15 p.m.: **Annual General Meeting and Clinico-Pathological Demonstation.**

WEST OF ENGLAND CHILD HEALTH GROUP

Secretary: Dr. P. G. Roads, Central Health Clinic, Tower Hill, Bristol 2

Meetings at the Children's Hospital, Bristol.
21st Oct., at 8.30 p.m.: Miss Doris Odlum, M.A., M.R.C.S., D.P.M., **"Adolescence".**
6th Nov., at 8.30 p.m.: E. Rogan, Esq., L.R.C.P.I., D.P.H., **"Age of the Menarche".**
9th Dec., at 8.30 p.m.: Professor A. V. Neale, **"Paediatrics in the South Pacific".**
15th Jan., at 8.30 p.m.: Professor A. I. Darling, **"Preventive Dentistry".**
5th Feb., at 5.30 p.m.: David M. Stevens, Esq., M.B., F.R.C.S., D.L.O., **"Catarrhal Deafness in Childhood".**
12th Mar., at 8.30 p.m.: P. R. Evans, Esq., M.D., M.SC., F.R.C.P., **"Baby Farming".**
May (date to be announced later): Miss Cecile Hopkins, M.R.C.S., L.R.C.P., D.P.H., **"Problem Families".**

B.M.A. (Barnstaple Division)

Secretary: Dr. S. G. Brook, Eddleston, Park Lane, Barnstaple

1st Oct., at 8.30 p.m.: **Clinico-Pathological Meeting** at the North Devon Infirmary, arranged by Dr. R. Tierney. (Preceded by supper at 7.30 p.m.)
1st Nov., at 8.30 p.m.: H. L. Shepherd, Esq., CH.M., F.R.C.O.G. and G. R. Airth, Esq., M.B., B.S., D.M.R.D., **"The Use of X-rays in Pregnancy",** at the North Devon Infirmary. (Preceded by supper at 7.30 p.m.)
Dec. (on a date to be arranged): A Farewell Dinner to S. C. Shaw, Esq., F.R.C.S., on his retirement.
Other meetings yet to be arranged include a Clinical Meeting at Bideford Hospital, an address on "Modern Therapeutics" and a Cocktail Party to welcome new members.

B.M.A. (Bristol Division)

Secretaries: Dr. A. S. Anderson, 1 Dean Lane, Bristol 3, and Dr. Sarah Walker, 44 Kewstoke Road, Bristol 9

7th Oct., at 8 p.m.: Donald Hunter, Esq., C.B.E., M.D., F.R.C.P., **"New Problems in Public Health"** (Sir Charles Hastings Lecture 1958), Victoria Rooms. This lecture is open to the general public.

19th Oct., at 2.45 p.m.: St. Luke's Tide Service in Bristol Cathedral. Preacher: The Very Rev. D. E. W. Harrison, Dean of Bristol.

21st Nov.: **Annual Dance,** Grand Spa Hotel, Clifton.

21st Jan., at 8.30 p.m.: Allan F. Rogers, Esq., M.D., **"The Antarctic Expedition".** At the Royal Fort.

6th May, at 8.30 p.m.: **Annual General Meeting** at the Royal Fort.

27th May, at 8.30 p.m. (provisional date): **Clinical Meeting** at Southmead Hospital.

12th June, at 4 p.m. (provisional date): Reception for newly qualified doctors. In the Garden Hall, the Royal Fort.

B.M.A. (Cornwall Division)

Secretary: Dr. E. Townsend, Fieldways, Tregenna Lane, Camborne

1st Nov., at 2.30 p.m.: **Clinical Meeting,** Royal Cornwall Infirmary, Truro.

6th Nov., at 8.30 p.m.: H. Osmond-Clarke, Esq., C.B.E., F.R.C.S., **"Pain in the Head and Neck".** (B.M.A. Clinical Lecture.) Royal Cornwall Infirmary, Truro.

27th Nov.: **Annual Dinner and Dance,** Hotel Bristol, Newquay.

Apr. (details to be announced later): Sir Stanford Cade, K.B.E., C.B., F.R.C.S. Lecture on Radiology.

B.M.A. (Gloucestershire Branch)

Secretary: Dr. H. G. Scott-Kerr, 29 Leckhampton Road, Cheltenham

Cheltenham meetings at the General Hospital and *Gloucester* meetings at Hut 6, City General Hospital, Great Western Road.

9th Oct.: R. Harvey, Esq., M.B.E., F.R.C.S., **"Doctor in Prison".** Presidential Address, Cheltenham.

13th Nov.: **Clinical Meeting,** Gloucester.

11th Dec.: D. B. E. Belson, Esq., Director of John Harvey & Sons, Ltd., Bristol, **"Wines and the Palate".** Film: "Talk and Tasting", Cheltenham.

8th Jan.: R. Belsey, Esq., M.S., F.R.C.S. will read a paper. Gloucester.

12th Feb.: **Clinical Meeting,** Cheltenham.

12th Mar.: H. Mower, Esq., M.R.C.S., L.R.C.P., D.L.O., **"Deafness",** with film of demonstration of cases. Gloucester.

9th Apr.: Sir Gordon Gordon-Taylor, K.B.E., C.B., M.D., F.R.C.S., **"In Retrospect".** B.M.A. Lecture, Cheltenham.

7th May: **Annual General Meeting.** F. B. P. Evans, Esq., M.B., B.S., **"Fads, Prejudices and Errors in General Practice".** Tewkesbury.

11th June: Address by President's guest. (This is in place of Mr. Stallard's paper which will not now be given.)

B.M.A. (South-Western Branch)

Secretary: Dr. F. E. Graham-Bonnalie, St. John's House, 37 The Strand, Topsham, near Exeter

30th Oct., at 8.15 p.m.: Address by A. Lawrence Abel, Esq., M.S., F.R.C.S., Royal Devon and Exeter Hospital.

1st Nov., at 2.30 p.m.: **Clinical Meeting.**

BRISTOL SOUTH MEDICAL GROUP

Secretary: Dr. H. I. Howard, Claremead, 102 Church Road, Bishopsworth, Bristol 3

Meetings take place on the first Sunday in each month during the winter at the Bristol General Hospital at 8.p.m.

COSSHAM MEDICAL SOCIETY

Secretary: Dr. R. C. Wyldesley, 267 Soundwell Road, Kingswood, Bristol

Meetings at Cossham Hospital at 8.45 p.m.

14th Oct.: R. M. Courtney, Esq., M.B.CH.B., D.P.H. **Presidential Address.**

4th Nov.: R. T. Routledge, Esq., M.B., F.R.C.S., **"A Tale of Two Cities".**

2nd Dec.: R. Wohlfeld, Esq., M.D., **"Osteopathy—Pro and Contra".**

6th Jan.: Professor C. Bruce Perry, **"Iatrogenic Diseases".**

3rd Feb.: Donal Early, Esq., L.R.C.P.I., D.P.H., **"Anxiety States and their Treatment".**

3rd Mar.: J. A. Cosh, Esq., M.D., M.R.C.P., **"The Management of Hypertension".**

17th Mar.: **Annual Dance,** Ashton Court Country Club.

7th Apr.: **Annual General Meeting.**

BATH CLINICAL SOCIETY

Secretary: Dr. J. R. Bolton, West Lodge, 61 Englishcombe Lane, Bath

All meetings will be held at 8 p.m. at the Royal United Hospital, Bath, except the November meeting which will be held at St. Martin's Hospital, Bath.

10th Oct.: H. Lovell Hoffman, Esq., M.D., F.R.C.P. and J. Montgomerie, Esq., M.B.CH.B., F.F.A.R.C.S., **"Management of Respiratory Complications of Neurological Diseases".**

14th Nov.: **Clinical Meeting.**

12th Dec.: Mrs. C. D. Cross, M.D., M.R.C.P., **"Treatment and Prognosis in Haemolytic Disease of the Newborn".**

9th Jan.: J. A. Cosh, Esq., M.D., M.R.C.P., C. P. Sames, Esq., M.S., F.R.C.S., A. C. P. Thomson, Esq., M.B.CH.B., D.M.R., G. H. Williamson, Esq., M.B.CH.B., **Symposium on Disorders of the Colon.**

13th Feb.: F. Kohn, Esq., M.D., **"Ignaz Semmelweis".**

13th Mar.: Norman Capon, Esq., M.D., F.R.C.P. Guest Speaker.

10th Apr.: **Clinical Meeting.**

25th Apr.: **Annual Dinner** at Fortt's Restaurant.

CORNWALL CLINICAL SOCIETY

Secretaries: Dr. J. D. Hardy, Royal Cornwall Infirmary, Truro, and Dr. B. A. Gwynne Jenkins, Central Chest Clinic, Tuckingmill, Cornwall

2nd Oct.: H. Osmond-Clarke, Esq., C.B.E., F.R.C.S., **"Pain in the Neck and Arm".** (B.M.A. Lecture.) Royal Cornwall Infirmary.

7th Nov.: **Clinical Meeting.** Camborne–Redruth Hospital.

27th Nov.: **B.M.A. Dinner and Dance.** Hotel Bristol, Newquay.

4th Dec.: **Clinical Meeting or Symposium.** Royal Cornwall Infirmary.

9th Jan.: Short Papers by General Practitioners, Camborne–Redruth Hospital.

6th Feb.: Sir William Porter MacArthur, K.C.B., D.S.O., O.B.E., **"Samuel Pepys and the London Plague".** Royal Cornwall Infirmary. (Ladies' Night.)

6th Mar.: **Clinical Meeting,** Chest Hospital, Tehidy.

Apr. (date to be announced later): **B.M.A. Lecture.** Royal Cornwall Infirmary.

7th May: **Cases and Demonstrations** by the Consultant Staff, Royal Cornwall Infirmary.

5th June: J. G. Hastings Ince, Esq., M.B., B.S., F.R.C.O.G. **Presidential Address. Annual General Meeing,** Camborne–Redruth Hospital.

All meetings at 8 p.m.

ROYAL DEVON AND EXETER HOSPITAL POSTGRADUATE PROGRAMME

Secretary: Dr. Anthony Daly, 30 Barnfield Road, Exeter

The Exeter Postgraduate Meetings will take place on Tuesdays at 8.30 p.m. from 14th October to 9th December.

SOCIETY OF MEDICAL OFFICERS OF HEALTH WEST OF ENGLAND BRANCH

Secretary: Dr. R. H. G. H. Denham, Bathavon Council Offices, 30 Westgate Buildings, Bath

The Society is divided into two sections, Northern and Southern. Each section holds four meetings a year, one of which being a joint meeting of both sections.

Northern Section:

4th Oct.: D. E. Cullington, Esq., M.A., M.B., B.CHIR., D.C.H., D.P.H. **Presidential Address.** At Taunton.

3rd Jan.: At Bristol (details not yet available).

4th Apr.: At the Pump Room, Bath (details not yet available).

May: Joint Meeting of Northern and Southern sections (details not yet available).

SOUTH SOMERSET MEDICAL CLUB

Secretary: Dr. N. K. Dryden, 2 Penn Hill, Yeovil

16th Oct.: F. S. W. Brimblecombe, Esq., M.D., M.R.C.P., **"Common Problems in Paediatrics".**

20th Nov.: **Clinical Meeting.**

Jan. (date to be announced later): **Dinner and Dance.**

19th Feb.: **Paper** by J. F. Davidson, Esq., O.B.E., M.B.CH.B., D.P.H.

19th Mar.: **Symposium.**

18th Apr.: **Paper** by T. S. Wilson, Esq., M.D.

TORQUAY AND DISTRICT MEDICAL SOCIETY

Secretaries: Dr. D. K. MacTaggart, " Broadwater", Preston Down Road, Paignton, and Dr. I. F. R. Sutherland, 1 Shiphay Avenue, Torquay

Meetings are held at Torbay Hospital at 8.30 p.m. unless otherwise stated.

30th Oct.:
4 p.m. R. S. Corbett, Esq., M.CHIR., F.R.C.S., **"Ulcerative Colitis".** Torbay Hospital.
7.30 p.m. **Dinner and Dance** at the Palace Hotel, Torquay.

13th Nov.: Maurice Campbell, Esq., O.B.E., F.R.C.P., **"Treatment of Coronary Disease".**

19th Dec.: Allan F. Rogers, Esq., M.D., **"The Trans-Antarctic Expedition".**

29th Jan.: A. J. Rook, Esq., M.D., M.R.C.P., **"Eczema in Infancy".**

26th Feb.: Murray A. Falconer, Esq., M.CH., F.R.C.S., **"The Treatment of Malignant Disease by Hypophysectomy".**

12th Mar.: **Clinical Discussion—"Infertility"** opened by Mrs. Margaret Jackson, M.B., D.OBST., R.C.O.G.

30th Apr.: A. S. King, Esq., B.SC., PH.D., F.R.C.V.S., **"Diseases of the Invertebral Disc".** Joint meeting with the Western Counties Veterinary Association.

28th May: **Annual General Meeting.** Victoria Hotel, Torquay.

WEST PENWITH MEDICAL SOCIETY

Secretary: Dr. Eileen E. Wood, West Cornwall Hospital, Penzance

16th Oct., at 8.15 p.m. **Annual Dinner.** Queen's Hotel, Penzance.

20th Nov.: T. S. Wilson, Esq., M.D., **"Restlessness and Mental Confusion in the Elderly".** Mrs. E. E. Wood, M.R.C.S., L.R.C.P., **"Anaemia in Old Age".**

18th Dec.: J. H. Willis, Esq., of Melbourne Botanical Gardens. **"The Australian Flora".** Illustrated by colour photographs. This is an open meeting to members' wives and friends.

15th Jan.: Mark Hewitt, Esq., M.B., B.S., M.R.C.P., **"Dermatological Problems and Cases".**

19th Feb.: R. Adlington, Esq., F.R.C.S., **"Vascular Emergencies".**

19th Mar.: **"Laboratory Aids to Diagnosis"** by the staff of West Cornwall Hospital Laboratory.

16th Apr.: **B.M.A. Lecture** by Sir Stanford Cade, K.B.E., C.B., F.R.C.S. Jointly with Cornwall Clinical Society and Cornwall Division of the B.M.A., Royal Cornwall Infirmary.

21st May: **Medico-Legal Session.**

18th June: **Clinical Meeting.**

Notes and News

BRISTOL MEDICAL DRAMATIC CLUB

The next production will be " Dr. Angelus " by James Bridie at the Y.W.C.A., 30th Oct.–1st Nov. Tickets (5/– and 3/6) may be obtained from Dr. P. B. Bailey, Whitchurch. Cast: James Hope-Scott, Eve Borland, Roger Carr, Joy Loxton, John Naish, Kenneth Malcolmson, Anne Malone, Julian Benson, Christopher Hallett.

WESSEX RAHERE CLUB

Secretary: Mr. A. Daunt Bateman, 11 The Circus, Bath

1st Nov.: **Dinner** at Grand Spa Hotel, Bristol. G. Bodley Scott, Esq., D.S.O., M.R.C.S., L.R.C.P., will be the guest of honour.

Membership of the Club is open, without subscription, to all Barts. Graduates practising or resident in the West Country.

PHYSIOTHERAPISTS' CONGRESS

This year the Chartered Society of Physiotherapy held its Annual Congress in Bristol. The programme extended over three days, 11th–13th September and much of the intellectual fare was provided by local talent.

The lectures, which were delivered to an appreciative audience of over 400 included one by Mr. R. H. R. Belsey about the dangers and prevention of lung cancer, from which a quotation found its way into *The Observer's* "Sayings of the Week". Mr. H. K. Lucas gave a stimulating account of modern management of the handicaps due to poliomyelitis and dwelt in detail on aids and advice for the paralysed housewife. Mr. K. H. Pridie discussed the surgical repair of arthritic knees—a stiff painless knee is more useful than a bent or painful one—and a physiotherapist should educate the patient how to adapt himself to this compromise.

Dr. R. C. Wolfinden suggested tackling the ever-mounting toll of coronary artery disease by diet and exercise. He also advocated wider use of home care: patients preferred it, money would be saved, and hospital beds used to better purpose.

The entertainments included visits to various Medical show-pieces with a tantalizing counter-attraction at the same time of a visit to the Wine Cellars of Messrs. Harvey. There was either a reception or a dinner each evening. In after-dinner speeches Mr. A. L. Eyre-Brook talked eruditely about the history of Bristol, and Mr. Norman Capener applauded the value of actual massage itself and enjoined doctors and physiotherapists to co-operate closely for the benefit of the patient.

PRIZES

L. E. Attenborough Silver Gilt Medal: B. L. Dahl.
L. E. Attenborough Bronze Medal: G. S. Jones.
Tibbits Memorial Prize: M. Nicholas.
David Rayner Prize in Gynaecology: Alison J. McConnell.
Green-Armytage Prize in Obstetrics and Gynaecology: R. A. Sharpe.
Gold Medal in Child Health and Paediatrics: Susi G. Leyton.

EXAMINATION RESULTS

M.R.C.S., L.R.C.P.: T. O. Dada, Glenys R. Packer, E. O. Olurin.

Appointments

UNITED BRISTOL HOSPITALS

Name	*Appointment*	*Formerly*
VOWLES, K. D. J., M.B.(BRISTOL), F.R.C.S.	Lecturer in Surgery, University of Bristol. Hon. Senior Registrar.	
WRIGHT, D. H., B.SC., M.B., CH.B.(BRISTOL)	Research Assistant, Registrar grade.	

H. Summers, M.B.(Manch.), M.SC.(Bristol) has been appointed Assistant M.O. Mid-Cheshire area.

R. J. K. Tallack, M.B.(Bristol), D.P.H., has been appointed Assistant M.O.H. and Schoo M.O., Westmoreland.

D. M. M. Thomson, M.B.(Bristol), has been appointed M.O., Uganda.

SOUTH WESTERN REGIONAL HOSPITAL BOARD

July–August, 1958

BRISTOL

Name	*Appointment*	*Formerly*
PAGE, F. T., M.D.(LOND.), M.R.C.P.(LOND.).	Consultant Physician with an interest in Neurology to the Bristol Clinical Area.	Senior Medical Registrar, Central Middlesex Hospital.
LESLIE, R. F., M.B., B.S.(LOND.).	Registrar to Bristol Chest Clinic and associated hospitals.	House Physician, Tehidy Hospital, Camborne.
MATTS, S. G. FLAVEL, M.B., CH.B.(B'HAM), M.R.C.P.(EDIN.).	Medical Registrar, Southmead Hospital.	Senior R.M.O. and Medical Asst. to Neurosurgical and Thoracic Surgical Depts., Frenchay Hospital.
FLOOD, R. A., M.B., CH.B.(ST. ANDREWS).	Registrar, Bristol Mental Hospital Group.	Senior House Officer, Barrow Hospital.
SPEAR, F. G., M.B., CH.B. (BRISTOL), D.P.M. (CONJOINT)	Registrar, Bristol Mental Hospital Group.	Medical Officer, Royal Air Force.
NABAR, B. V., M.S. (BOMBAY), F.R.C.S. (EDIN.).	Surgical Registrar, Southmead Hospital.	Surgical Registrar, Oldham and District General Hospital.

NORTH GLOUCESTERSHIRE

Name	*Appointment*	*Formerly*
CHAKRABARTI, R., M.B. (CALCUTTA).	Medical Registrar, Gloucestershire Royal Hospital, Gloucester.	Medical Registrar, Grimsby General Hospital.

BATH

Name	*Appointment*	*Formerly*
WILLIS, A. J. P., M.B., B.S. (LOND.).	Medical Registrar, Bath Group of Hospitals.	S.H.O. in Pathology, Mile End Hospital, London.

DEVON AND CORNWALL

Name	*Appointment*	*Formerly*
PICKERING-PICK, M. E. M.B., B.S. (LOND), D.A.	Anaesthetic Registrar, South Devon and East Cornwall Hospital, Plymouth.	S.H.O. Anaesthetist, South Devon and East Cornwall Hospital, Plymouth.
MANSFIELD, G. C., M.B., CH.B. (GLASGOW), D.R.C.O.G., D.A.	G.P. Anaesthetist, South Hams Hospital, Kingsbridge.	Is in general practice in Totnes and G.P. Anaesthetist at Paignton and District Hospital.
BAILEY, L. J., L.D.S., R.C.S.	S.H.D.O., Newton Abbot Hospital.	In practice in Newton Abbot.
DONOVAN, J. F., M.R.C.S., L.R.C.P., D.P.M.	Medical Superintendent, St. Lawrence's Hospital, Bodmin.	Consultant Psychiatrist and Physician Supt., Broadgate Hospital, Beverley, Yorks.
DHANDA, S. P., M.B., B.S. (MADRAS).	Surgical Registrar, South Devon and East Cornwall Hospital, Plymouth.	Surgical Registrar, Workington Infirmary.
LLEWELYN, D. M., M.B., B.S. (SYDNEY).	Surgical Registrar, Royal Cornwall Infirmary, Truro.	Orthopaedic Registrar, Hammersmith Hospital.

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